Menopause

Daniel R. Mishell Jr, MD

The decline of ovarian function occurs gradually, and the cessation of menses is only one facet of the climacteric process. After the menopause, ovarian production of estrogen ceases and circulating levels fall dramatically.

Decreasing estrogen production leads to atrophy of the vagina, which can produce the distressful symptom of senile vaginitis or atrophic vaginitis. Estrogen deprivation also can cause the structures that support the uterus, the cardinal and uterosacral ligaments, to lose their tonicity and uterine descensus could occur. The trigone of the bladder and the urethra are embryologically derived from estrogen-dependent tissue, and estrogen deficiency can lead to their atrophy, producing symptoms of urinary urgency incontinence, dysuria, and urinary frequency. Another problem that can develop with decreased circulating estrogen levels is decreased synthesis of collagen that forms the connective tissue beneath the vaginal/epithelium. This change may decrease the support of the posterior urethro-vesicle angle and urinary stress incontinence can develop. Loss of collagen also can lead to clinically symptomatic cystoceles and/or rectoceles. These urinary symptoms can be alleviated or prevented with estrogen replacement therapy.

The pathognomonic symptom of menopause is the hot flush or flash, which is caused by a decrease in circulating estrogen levels. About 75% of all women going through menopause develop hot flushes. These flushes frequently occur at night, awaken the individual, and then produce insomnia. Hot flushes do not persist in most women for more than 2 to 3 years, and it is uncommon for a woman to have hot flushes that last more than 5 years after menopause. The most effective treatment for the hot flush is estrogen. Since so many of the hot flushes occur at night, it is advisable for the patient to ingest the estrogen tablet before bedtime.

Several studies have demonstrated that estrogen improves many psychological symptoms significantly better than placebo, particularly depression, in addition to relieving the hot flush and allowing the patient to sleep better. Estrogen users are less likely to develop dementia and Alzheimer's disease than non-estrogen users. Postmenopausal estrogen users have significantly thicker skin and a greater amount of collagen in the dermis than non-estrogen users. Systemic estrogen use can retard wrinkling and thinning of the skin postmenopausally.

Osteoporosis is defined as an asymptomatic reduction in the mass-per-unit bone volume (density) so that there is a significantly increased risk of fracture in the absence of trauma. Postmenopausal osteoporosis initially affects trabecular bone that is present in the vertebral column and distal radius. Osteoporosis develops more slowly in cortical bone, which is present in the limbs. About 1% to 2% of bone mass is lost each year after menopause. Osteoporosis is an asymptomatic disease, and its presence usually is not detected until a fracture occurs many years later. At least 25% of the bone needs to be lost before osteoporosis is diagnosed by routine x-ray examination, so other procedures such at CT scan or DEXA are used to detect this disorder.

In women undergoing a normal menopause, fractures begin to occur about age 60 in structures composed mainly of trabecular bone. By age 60, 25% of Caucasian and Oriental women develop spinal compression fractures. Loss of bone mass in cortical bone occurs at a much slower rate, so osteoporotic fractures of the femur usually do not begin to occur until about age 70 or 75. Patients with osteoporosis have a higher bone resorption rate than normal. With a few months of estrogen treatment, bone resorption rates returned to normal. Bone formation in patients with osteoporosis is normal before and after the estrogen therapy. The best way to prevent loss of calcium from bone in postmenopausal women is to administer exogenous estrogens.

Epidemiologic studies have shown that estrogen therapy reduces the amount of postmenopausal bone loss as well as the incidence of fracture. The minimum dosage of estrogen needed to prevent osteoporosis is 0.625 mg of conjugated equine estrogens, 0.625 mg of estrone sulfate and 0.5 mg of micronized estradiol. Ingesting the recommended daily intake of dietary calcium (800 mg to 1000 mg/day) during the adolescent years results in greater peak adult bone mass than occurs if insufficient calcium is ingested. Thus, postmenopausally with steady loss of bone, women who when premenopausal ingested the recommended intake of calcium are less likely to have sufficient reduction in bone density to cause fractures than those ingesting insufficient calcium. Dietary calcium supplementation without estrogen or weight-bearing exercise do not prevent postmenopausal bone loss.

In contrast to the increase in blood pressure that has been reported in some women using oral contraceptives, no such increase has been observed with use of estrogen replacement therapy. It is safe to prescribe estrogen replacement for post-

Daniel R. Mishell Jr, MD
The Lyle G. McNeile Professor and Chairman
Department of Obstetrics and Gynecology
University of Southern California School of Medicine
Los Angeles, California

menopausal women with hypertension, and if blood pressure increases while they are receiving this treatment, it is unlikely that the estrogen is the cause of the blood pressure elevation. There is no epidemiologic evidence of an increased incidence of thrombophlebitis or thromboembolism in postmenopausal estrogen users as compared with control subjects.

An abundance of epidemiologic data indicates that estrogen replacement therapy retards the development of atherosclerosis in postmenopausal women and reduces their risks of developing myocardial infarctions and cerebrovascular accident (CVA, stroke) by about 50%. The main mechanism whereby oral estrogen replacement retards atherosclerosis postmenopausally is prevention of the adverse alterations in endogenous circulating lipid levels that normally occur after the menopause, but estrogen also acts directly on the arterial system to improve blood flow.

Much concern has been raised about the neoplastic risks of postmenopausal estrogen replacement therapy, particularly breast and endometrial cancer, since these areas are estrogen target tissues.

The possibility exists that estrogen can stimulate a nonpalpable breast cancer, and carcinoma of the breast can exist in the preclinical state for as long as 8 years before it is palpable. Therefore it is advisable to obtain a mammogram to rule out subclinical breast cancer on all patients before initiating estrogen therapy and annually thereafter.

Many epidemiologic studies have investigated the relation of exogenous estrogen and the incidence of breast cancer. The epidemiologic data generally are reassuring as most studies show no increased risk of development of breast cancer among postmenopausal estrogen users, with the possibility of a slightly increased risk with long-term use. At present, it is not clear what effect if any the addition of a progestin to estrogen replacement therapy has on the risk of breast cancer.

Many epidemiologic studies have reported there is significantly increased risk of endometrial cancer developing in postmenopausal women who are ingesting estrogen without progestins as compared with non-estrogen users. The risk increases with increasing duration of use of estrogen and with increasing dosage. The endometrial cancer that develops in estrogen users is nearly always well differentiated and is usually cured by performing a simple hysterectomy. The risk of developing endometrial carcinoma for women receiving estrogen replacement can be markedly reduced by giving progestogens. The use of progestins lowers the chances of postmenopausal estrogen users' developing cancer of the endometrium, and therefore progestins should usually be given to postmenopausal women receiving estrogen if they have a uterus. The addition of a progestin to estrogen therapy does not appear to cause an increase of any other systemic disease and acts synergistically with estrogen to cause a slight increase in bone density. The use of synthetic progestins, however, may reverse the beneficial effect of estrogen upon serum lipids.

One of the primary reasons that postmenopausal women decide not to use estrogen, or discontinue its use, is the occurrence of uterine bleeding. For this reason, combination instead of sequential estrogen-progestin regimens are being increasingly prescribed as the former regime is usually associated with no bleeding after the first few months. For women without a uterus it is unnecessary to add a progestin.

Domestic Violence—A Medical Perspective
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4,000 women are killed in the U.S. each year by their current or former partners, with the great majority of these murders occurring after the woman has ended the relationship. During the 1980s alone, an estimated 50,000 American women died as a result of domestic violence. Incredibly, this figure nearly matches the number of U.S. fatalities for the Vietnam war.

How are we as a society to respond to this new understanding of domestic violence as a medical crisis? One way to begin is by providing training for health care providers and insisting that medical schools include domestic violence education in their curriculums—medical providers have the potential to reach countless victims. Since almost all battered women must occasionally see family doctors, pediatricians, dentists, counselors, or others in the health care field, opportunities abound to recognize the indicators of abuse and to make referrals to life-saving resources.

In order to heighten the awareness of the medical community, and society at large, however, we must begin as individuals to shed our own heavy denial, which protects us from facing the true enormity, severity, and randomness of domestic violence. We must shatter our outdated stereotypes about why abuse occurs once and for all, and exactly who "those people"—victims and batterers—really are. In addition, we must stop blaming the victim by constantly asking, "Why does she stay?" and instead refocus our attention and indignation on the person using the violence by asking, "Why does he hit?"

Coming to terms with any unpleasant social truth is an uncomfortable experience. To let go of denial and grasp, for perhaps the first time, the truly devastating ramifications of domestic violence is especially disturbing. There is no going back. But until we as individuals are willing to risk some degree of personal discomfort in exchange for the truth, we will be robbing ourselves of golden opportunities to help many victims—perhaps even our own co-workers, neighbors, friends, sisters, daughters, or mothers—who may someday tell us that their injuries are from a "fall down the stairs" or from "playing volleyball."

Editor's Note
In 1989, after surviving a brutal attack in which she was beaten and stabbed by her estranged husband, Julie Owens left her career in special education and devotes her full-time efforts to the field of family violence. She gives domestic violence training seminars to professionals and trains officers and counselors in the Honolulu Police Department's DART program (Domestic Abuse Response Team).